THE NERVOUS COMPLICATIONS AND SEQUELÆ OF SMALLPOX.*

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Smallpox, like other infectious diseases, may present various nervous complications and sequelæ. Indeed, almost the first effect of the variolous poison is observed in the violent frontal headache, which constantly is an early symptom. Sharply contrasted with the many other complications and sequelæ of variola, the affections of the nervous system are not strictly confined to certain stages of the disease itself, not necessarily immediately sequential, nor are they at all related to the severity of the infection. It seems that the variolous process possesses, in a marked degree, the power to awaken latent predispositions to nervous disease, as well as to independently disorder function and create anatomical alterations in the various nervous structures of the body. These affections may be gross anatomical, purely functional, or both. Many cases present a varied clinical picture, which leaves us in doubt as to the exact nature of the lesion, and until further post-mortem examinations and other investigations have bettered our present knowledge, we must be content with collecting the rare and interesting cases for the literature and for future investigations and diagnosis. These complications and sequelæ invade all possible provinces of the nervous system—the brain, spinal cord, and peripheral nerves. Truly, the nervous complications and sequelæ of this loathsome disease offer a most fruitful and little-worked field for research.

Convulsions are often seen in children, and usually precede the eruption. They are mentioned by Trousseau,[1] who endorses the belief of Sydenham,[2] that they possess diagnostic value, especially in the young. Sydenham[3] states that convulsions occur more frequently in smallpox than in any of the xanthematous diseases.

Smallpox often exerts a profound effect upon the pysche.

In order of frequency, as a cause of the febrile deliria and psychoses, smallpox is placed sixth by Berkley.[4] Kraeplin,[5] who is, perhaps, the greatest authority upon this subject, also places variola in the same causative relation.

We may recognize at least four types:

1. Initial delirium.
2. Febrile delirium.

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3. Collapse or exhaustion delirium.
4. True postvariolous insanity.

The occurrence of initial delirium is quite constant, usually appearing toward the evening of the second or third day; the mind wanders, the speech becomes incoherent, and a true confusional condition develops. Throughout the attack there is much sleeplessness and great disquietude. This delirium differs entirely from that with specifically colored delusions and accompanied by tremors, the delirium alcoholicum, which commonly occurs in inebriates attacked by smallpox.

Seppilli and Maragliano[6] reported three cases of acute mania occurring in smallpox. Two recovered and one remained incurably insane. Trousseau[7] referred to the case of a woman who was seized with an acute mania during the progress of a mild attack of smallpox. She had never before shown any symptoms of mental disturbance. Corlett[8] records the occurrence of an acute mania in the course of a discrete smallpox. The sufferer remained violently insane for six days, when death came.

Grave changes in the mental state, with peculiar speech disturbances, have been observed to follow variola by Welch,[9] Westphal,[10] Otto,[11] Foville,[12] Jaccoud,[13] Riva,[14] Quinquaud,[15] Leudet,[16] Whipham and Myers,[17] Pforzheim,[18] Béhier and Lionville,[19] Long,[20] the writer,[21] and others. These mental changes seem to be a part of widespread changes in the nervous system, and yet sufficiently uniform in their symptom groups and their pathological pictures to deserve separate and detailed description, which will be given later, in connection with two personally observed cases.

The occurrence of true dementia paralytica has been observed to follow variola by Mabille[22] and others. In fact, the writings of Hoppe,[23] Lagardelle,[24] Berthier,[25] Kiernan,[26] and many lesser known observers show that smallpox is a fruitful cause of mental alienation.

Meningitis, fortunately, is a rare complication. It is usually purulent, and probably a metastasis from the skin lesions. Gregory[27] states that it is most liable to occur in children and during the period of suppuration and beginning desiccation.

Reiner[28] has recorded a severe meningitis of the convexity occurring as a variolous complication.

The occurrence of paralysis in smallpox has been commented upon since that exanthem's history passed from tradition to record. Indeed, literature bears out the statement of Landouzy,[29] that "of all the eruptive fevers smallpox is, without contradiction, the one in which paralysis most frequently occurs, either at the onset, in the course of, or at the decline." How frequently it occurs, unfortunately, we have no means of ascertaining. That smallpox is less liable to cause the complication than typhoid fever seems to be the opinion of Landouzy,[30] but from a limited personal observation and an extensive search of the literature, I question the statement.

The nature, origin, and seat of the various paralytic affections attending and consequent to variola are so varied that classification is most difficult. That they may originate in the brain, spinal cord, or peripheral nerves is certain. It is also positive that we may have a combination of two or more of these forms.

Paralysis may occur at the very onset of the infection, at any time during its course, or at any period of convalescence.

The majority of the paralytic affections are, as in other acute infections, of neuritic origin. Landouzy[31] believed that paraplegia was most frequent. I believe, however, that he included the cases of brachial paraplegia, as well as the crural monoplegias, in his consideration. Rejecting these as most likely peripheral neuritides, we place paraplegia of spinal origin farther down in the list of nervous complications and sequelæ of variola.

All are agreed upon the rather fortunate outcome of most of the paralyses of variola.

E. Wagner[32] has observed, on post-mortem examination, evidences of localized non-purulent encephalic disease. And that we may have a non-purulent meningoencephalitis of a simple character complicating variola, I believe, the first case to be reported in this paper will fully demonstrate.

Areas of simple softening and blood extravasations may occur in the brain and produce aphasia, monoplegia, or hemiplegia. According to Immermann,[33] such lesions may remain latent throughout life and be discovered only at autopsy.

Reiner,[34] Landouzy,[35] Edes,[36] Osler,[37] and many other writers mention and some relate personally observed variolous hemiplegias, but if the meagreness of the literature is an index of their infrequency, then they are rare, indeed.

Clinicians have long been aware of the relative frequency of cord affections in variola. The severity of the infection appears to have little to do with their occurrence, since they appear to develop with equal frequency in varioloid. The observed cases have been usually paraplegia, of the motor type, developing in any stage of the disease from the prodromal to a far-reaching period of convalescence. Leroy d'Etiolles[38] has recorded the occurrence of such affections in the stage of incubation before the proper beginning of the variola.

Trousseau[39] observed in cases where severe lumbar pain was complained of in the initial stage that a slight degree of paraplegia was often present. It was manifested by subjective numbness, tenderness, and loss of power. Occasionally the bladder was involved, producing difficult micturition and occasional retention. These symptoms usually disappeared with the appearance of the eruption, but in rare cases remained to the middle of the second week.

Henderson,[40] Westphal,[41] Kahler and Pick,[42] and others state that rarely acute ataxias have been observed to follow variola. It is quite probable that these cases of ataxia are ataxic forms of neuritis, although many of the cases of pseudomultiple scleroses manifested some ataxia. Henderson's[43] case of postvariolous ataxia presented numbness and tingling of the legs and hands. The skin reflexes and deep tendon reactions were all absent. The patient later recovered his power and station. In the discussion which followed Dr. Henderson's report, Dr. Whipham[44] referred to two similar cases, which, I presume, are embraced in the report of Whipham and Myers.[45] These cases have been looked upon as pseudotabes, the so-called ataxia variolique, but more likely belong to a class of disseminated focal inflammations scattered throughout the brain and spinal cord.

Peter Marie,[46] Sottas,[47] Long,[48] Charcot,[49] and others have observed typical examples of disseminated sclerosis following smallpox. The case of Sottas is quite typical, but it appears from the reports of most of these cases that, while for a time they presented an array of symptoms much like true disseminated sclerosis, they later slowly grew better, thus making a marked departure from the ordinarily progressive course of that disease.

Enough knowledge of the pathological changes has been found at the root of the disorder to demonstrate that we have disseminated encephalomyelitis which, although followed by a sequential sclerosis, has little or no tendency to extend or multiply itself. This subject will again be adverted to.

Westphal[50] recorded a case which was followed by paralysis of the legs and bladder that was due to disseminated foci of inflammation involving the gray and white matter of the spinal cord. Foci of softening in the structures of the cord have been described by Damaschino[51] and Joffroy,[52] Hayem[53] and Landouzy.[54]

Fiessinger[55] remarked the occurrence of acute myelitis appearing as a complication and disappearing with the disease.

Steven Mackenzie[56] has reported a case of poliomyelitis anterior as a sequel of variola. Damaschino and Roger[57] and Landouzy[58] record acute monoplegias in children presenting the clinical picture of acute inflammation of the cells of the anterior horns of the cord.

Although Huchard[59] observed not more than ten paraplegias in 2000 patients affected by smallpox, yet Landouzy[60] states that paraplegia is the most frequent form of variolous palsy.

A rapidly ascending myelitis presenting clinical symptoms of Landry's paralysis has been recorded by a number of authors: Marinesco and Oettinger,[61] Gubler,[62] Leyden,[63] Gros,[64] Chalvet,[65] Marie,[66] Bernhardt,[67] Gros and Beauvais.[68] These cases, as a rule, are rapidly fatal.

F. W. Goss[69] observed a typical case of acute ascending paralysis occurring early in a mild varioloid. It proved so quickly fatal that the patient died four days after the first paralytic symptoms.

Dr. M. Friedrich[70] personally related to me the details of a rapidly fatal case of undoubted ascending or Landry's paralysis, appearing in the stage of desquamation in a comparatively mild case of smallpox during our late epidemic in Cleveland.

It would appear from Oettinger and Marinesco[71] that the pathological picture of Landry's paralysis may exist in variola in combination with the disseminated myelitis described by Westphal. Indeed, a recent communication appears to have firmly established this fact.

Cases have been described by Vulpian[72] and others of acute ascending paralysis from variola in which a most careful search failed to show a myelitis. It seems reasonable to believe that these cases are polyneuritic in origin.

A careful examination of the literature convinces me that we have occurring as a complication or sequel of smallpox a disseminated encephalomyelitis, which possesses clinical features and pathological changes clearly entitling it to be recognized as a clinical entity, and which occurs with sufficient frequency to demand our special consideration and study. It occurs in other infectious diseases, as the case reported by Ebstein[73] conclusively proves. Ebstein's case is quoted particularly because it complicated typhoid fever and the symptom-complex was identical with that of the variolous cases, and the post-mortem disclosures were more complete and in keeping with the clinical findings.

I believe that these cases have in time past been described as and believed to be the disease which was named by Charcot disseminated sclerosis. Some of the reported cases bear a very close resemblance, but the majority are but coarse imitations. In this connection it is not thought wise to discuss the pros and cons of the question, but to report two personal observations.

I have been able to find fifteen cases in the literature, all presenting:

1. More or less ataxia in the four extremities, usually most marked in the legs.
2. Slowness and awkwardness of movement.
3. Slow, monotonous, explosive manner of speaking.
4. Faulty articulation.
5. A varying degree of mental degradation.
6. A decided tendency toward recovery.

The following personal observations are added to the literature:

To the courtesy of Dr. Martin Friedrich I am indebted for the privilege of examining and reporting this case:

Case I.—E. L., male, white, aged thirteen years, was normally born in the United States, and of German parents. He had never suffered any severe illness and was considered a large, well-developed boy. His father and mother are strong, healthy people, each giving a good family history. There are four other children in the family, all of whom are well. There have been no deaths in the family, nor history of miscarriages or stillbirths.

Edward was taken sick the last of June, 1902. The family attendant, Dr. Christian Sihler, was called and found the boy's temperature to be 103° F., from which it shortly rose to 104.5° F. Nothing could be found to account for the temperature, and because of numerous cases in the neighborhood, smallpox was suspected. On the fourth day an eruption appeared, and Dr. Martin Friedrich was called in consultation and made the diagnosis of discrete smallpox.

The temperature immediately fell on the development of the eruption, but little secondary fever developed, and the boy was not thought to be very ill. There had been no convulsions or other disturbances of the nervous system, either in the prodromal period or immediately following the eruption. He was sent to the Detention Hospital, where, for the first ten days, he was at no time confined to bed. He ate well, slept well, and did not complain. During the first week in the hospital an abscess developed on the right leg, which, although quite large, caused him little discomfort. He was homesick and anxious to go home, but was put off from time to time and felt very badly about the delay. On July 15th, and on the thirteenth day of his residence in the hospital, his father came to take him home, when the boy was discovered lying on the bed in a stupid condition and could not be aroused. It was thought, perhaps, that he was soundly sleeping, and the father went home without him. The attendant, failing to arouse the boy, informed Dr. Friedrich of his condition, who finally succeeded in awakening him, telling him to undress and get into bed. The boy recalls this and also that the nurse helped him to remove his clothing.

Dr. Friedrich states that his breathing was normal, but the pulse was very slow; the body was limp, with diminution of the reflexes. There were no changes in the pupils. The boy appeared dazed and his speech was slow and hesitating. The next day he was taken home. The stupor continued without any additional symptoms, except the advent of a temperature of 103.5° F. The abscess had been opened several days before this time and was discharging freely, but giving no pain. He remained in a stupor for a period of four or five days, during which time it was almost impossible to feed him; attempts to swallow were accompanied by choking, and it was necessary to feed him liquids with a spoon. From the hesitating speech he passed into a condition of complete aphasia, at the end of four days being unable to speak a word. The neck was stiff and the head slightly retracted; pulse very slow, rarely going above 60. There were no signs of ocular or other paralyses. The pupils were equal, of normal size, but reacted very slowly to various stimuli. The nose, larynx, and retinæ were carefully examined by Drs. Friedrich and Lenker and nothing abnormal discovered. No signs of thoracic or abdominal disease could be demonstrated.

His temperature continued irregularly elevated for a period of two weeks. His aphasia remained complete during this time, but he seemed able to understand everything that was said to him; could read print and writing, but he was not asked to attempt writing. There was no numbness, or paræsthesia, or loss of sensation. Before his aphasia became marked he complained bitterly of headache, and later, for a period of two weeks, by gestures continued to attempt to convey to his attendants the idea that the ache continued. If he attempted to get up on his feet he would fall, and he also fell out of bed and was unable to get up when down. After he was up and about his gait was markedly ataxic; he staggered like one intoxicated.

He cried a great deal, especially at the end of the second week, and seemed to be very much excited because he could not make his attendants understand what he wanted. Discovering that he cried because of hunger, his attendants began to feed him, when he developed almost a mania for food; would eat until they were afraid to feed him so much, and then shriek with rage and disappointment because he was not given more.

He was aphasic for a period of five weeks, and when speech returned by slow degrees it was distinctly scanning, slow, and thick. His parents relate that he was very much irritated by the presence of other children in the room and would usually order them out, speaking in a manner that was illustrated by the mother as follows: "You-u g-get r-i-i-ght o-u-ut—of-dis- ro-o-o-m—he-e-r-r"—this being the first formula which he seemed able to enunciate. The parents' and physician's account of this peculiar enunciation fits the apt description of Westphal, who said his patient's words came out as though each syllable was "squeezed out." His speech seemed to return rather rapidly; in fact, he soon insisted upon talking most of the time, but it was very hard to understand him, because of his enunciation being so thick, slow, and scanning.

I examined him in the following November: He is a large, well-developed boy, with hydrocephalic type of head. His eyes are prominent, pupils wide, vision normal; no nystagmus, ocular palsies, or pupillary disturbance; palpebral apertures are wide, showing the sclera above the cornea. His gait is peculiar. He walks like a boy who has on a pair of shoes that are too small for him. His station is not good. His parents state that if he receives a slight push he is unable to recover himself and falls. Knee-jerks are quick, no ankle clonus, no Babinski's sign. Left wrist-jerk and elbow-jerks are more marked than the right. His left hand is the seat of a peculiar movement resembling very closely an athetosis; this is particularly marked while walking. Indeed, his gait is worthy of attention and study. The legs are brought forward awkwardly and stiffly; the toes of both feet turn in, which his parents assure me was not the case previous to his illness. The toe of the left shoe is worn more than the right, and it is believed, when not on exhibition, that he drags this foot a little. The right hand hangs normally at his side, while the left is held partially behind him, the palm directed backward and on a line with the middle of the thigh, and at every step is the seat of a distinct athetoid movement involving the wrist and fingers. His parents inform me that this peculiar movement of the hand is often present while sitting still. All characteristics of chorea are absent, and I believe the movement to be an uncontrollable or athetoid movement consequent upon pathological alterations of the brain cells. He occasionally complains of headache.

There are now but few pits to be seen upon the face or body, and in a few months little will remain to show that he has suffered from smallpox. The only evidence of his aphasia at present is a certain thickness of speech and a tendency to stumble over syllables; in fact, he stumbles over the syllables in "United Irish Constabulary" as badly as a well-developed case of paresis. There appears to be, however, a slow and continuous improvement. His parents say that he seems to be as bright in school as before, but that his disposition has changed; that he is cranky and irritable. His father states that when children tease him about his tripping speech he weeps, "instead of punching them," as he formerly did when plagued.

There are some rather novel features in this case; some, indeed, suggesting hysteria; and were it not for the slow pulse, high temperature, and bulbar symptoms, one would be justified in suspecting a neurosis. The headache, slow pulse, and stupor point to an intracerebral pressure, probably due to a simple effusion. Assuming an intracerebral pressure, we have an explanation of the bulbar symptoms.

The aphasia, which was complete, and has left its mark in a halting speech, and an athetosis, point to an undoubted lesion of cerebral tissue, possibly hemorrhagic, or to areas of localized softening. The spinal symptoms of the case are as clearly marked as in Westphal's cases, and the clinical ensemble is certainly of that type.

CASE II.—A healthy young woman was taken sick with smallpox the last of May, 1902, and was removed to the Detention Hospital. While there she was under the care of Drs. A. B. Spurney and M. Friedrich, to whose courtesy I am indebted for most of the data of the case. She had a good family history and had been quite healthy herself, with the exception of frequent attacks of night terrors, which still continue.

After the usual prodromal symptoms the eruption appeared at about the fifth day, and, although discrete, was widely disseminated and the pocks large and close together. The secondary fever was not high, but the third day following the eruption she became very delirious. The delirium was not entirely characteristic of the ordinary febrile delirium, since it largely partook of the type of religious mania. Shortly after this delirium manifested itself she lost her speech, swallowing became difficult, and she lay helpless in bed; all four extremities were limp and perfectly useless. The urine and feces passed uncontrolled into the bed. Dr. Spurney states that the deep reflexes were absent at this time. Quite a number of abscesses developed, some of which persisted long into her convalescence. Her speech was almost entirely absent for a period of over three weeks, when it began to slowly return and had the same characteristics of the preceding case.

It was found when she was able to get out of bed that she was markedly ataxic, so much so that she reeled and staggered like a drunken person, and continued to do so for a period of about three months. The speech gradually returned, but her mental degradation was marked and interesting. She appeared and acted like an imbecile. She did not know enough to go to bed, but when sent to her room would lie down on the floor. She did not even know enough to dress herself, and had to be watched closely to prevent her undressing in public. She remained practically in this condition for two months, when a slight improvement began and continued progressively until one day, while in the chapel at prayers, she seemed to rather suddenly acquire her orientation, and from then on began to gain so rapidly that one month later she was able to go out to the Smallpox Hospital in Newburg and aid in the care of patients. Her improvement has been steady since then, and at the present time she has practically recovered all her faculties.

EXAMINATION. A recent examination reveals practically nothing, except, perhaps, a slight increase of the knee-jerks, a little slowness and tendency to syllabication of words. Her gait is not as good as before; a slight ataxic condition still obtains. The special senses are normal.

There seems to be evidence that peripheral neuritis occurs in smallpox with greater frequency than heretofore believed. It was formerly supposed that the few cases observed were of very limited extent and probably related to gross anatomical lesions of contiguous structures. Postvariolous paralysis of the soft palate and structures of the pharynx quite similar to the more frequent diphtheritic affections of those parts have been noted by Curschmann,[74] Leyden,[75] and others.

Combemale[76] has collected ten cases of peripheral neuritis occurring as complications or sequelæ of variola. In all of his cases some speech disorder was remarked, which he evidently believed to be due to palatal paralysis, or paralysis, or ataxia of the organs of phonation. Limited cutaneous anæsthesia and paralysis of single muscles, like the case of paralysis of the deltoid reported by Curschmann,[77] may rarely occur, and are certainly due to peripheral neuritis. Of great interest in this connection is the case of serratus magnus paralysis occurring during a variola observed by James J. Putnam.[78] This was undoubtedly due to a neuritis of the posterior thoracic nerve. Atrophy followed, and there was no recovery.

Vulpian[79] reported a case of paralysis and anæsthesia of the right circumflex, with weakness in the distribution of the same nerve on the opposite side.

Hitzig[80] has observed a case of double brachial neuralgia, with considerable loss of power; also neuralgia of one arm and shoulder, followed by paralysis of the deltoid alone. All of these cases appeared too early in the disease to be attributed to exhaustion or pressure from decubitus.

Eulenberg[81] has reported a case of partial paralysis of the left facial and both median nerves. The electrical reactions were little changed and recovery ensued.

Rosenblatt's[82] case of crural monoplegia was undoubtedly of peripheral origin, and, as before stated, it is my belief that Charcot's case of right brachial monoplegia, followed by permanent atrophy of the muscles, was also peripheral neuritis.

I find but one case of multiple neuritis following variola in which a post-mortem examination demonstrated conclusively the correctness of the diagnosis; it is reported by P. Grocco,[83] of Milan. The patient, a young man, six weeks after convalescing from smallpox developed all the symptoms of a multiple neuritis, later pneumonia, and finally died.

Gower[84] mentions a case of polyneuritis following varioloid. The patient died of pneumonia six months later, and the purely neuritic character of the paralysis and atrophy was demonstrated. Since he gives no reference to this case, I believe it to be the one reported by Grocco.

Joffroy[85] observed a patient who, while convalescing from a grave confluent smallpox, developed violent pains in the left arm and shoulder, which was followed by loss of power and muscular atrophy. Pulmonary tuberculosis supervened; the patient died, and a careful pathological and histological examination revealed a parenchymatous neuritis.

Charcot[86] has recorded a case of atrophy involving the muscles of the right arm consecutive to smallpox. It is possible that this case was a brachial plexus neuritis. Rosenthal,[87] of Vienna, reported a case of true progressive atrophy that appeared six weeks sequential to variola.

Indeed, the variolous poison or the combined infections which are so violently manifested in this disease may produce disturbed functions and actual degeneration of any part of the nervous system.

Functional nerve disturbances are not uncommon sequelæ to smallpox. They are commonly severe and persistent, but usually recover.

Epilepsy following variola has been mentioned by Bierlingus[88] as early as 1679.

Osler[89] states that rarely epilepsy appears during convalescence from smallpox.

H. E. Armstrong[90] relates a case of "catalepsy," with waxy mobility and afterward rigidity of the limbs and continuance of consciousness, followed by ecstasy, passing into obscene delirium," finally terminating in unconsciousness and death. The patient was a woman, aged twenty-four years, and in the tenth day of a severe variola, with confluent, crystalline eruption. * * *

Dreyfus-Brisac[91] observed an hysterical hemiplegia and hemianæsthesia of the right side disappear at the time of the efflorescence of a varioloid.

To illustrate the profound effects which smallpox may exert in the production of neurasthenia, the following case is related:

CASE III.—W. S., an unmarried policeman, born in the United States thirty-two years ago, gives a good family history, excepting the pertinent fact that he was born when his parents were at an advanced age. He states that his mother was fifty-four years old and his father sixty-four years old at the time of his birth. The patient suffered a sunstroke at sixteen years, and from that time has been troubled with frequent headaches. He says that while he has been physically strong, yet has had little endurance. He experienced a severe attack of measles at twenty-two years, and at twenty-eight years a violent attack of *la grippe,* and since has been visited by yearly attacks of the affection at about the same time each year.

Three and one-half years ago he suffered a violent attack of confluent smallpox, and is very badly pitted as a result. He was completely disabled for two months and then returned to light duty, but caught cold and was laid up two months longer, suffering at this time rheumatic pains and remaining unexplainably weak. He again returned to work, when he suffered further relapse of pain and weakness and general indisposition. From then until two years ago, when the final collapse came, he was rarely able to be on light duty more than a week at a time. Shortly before this collapse he had an attack of severe pain in the left upper chest, which extended down the arm to the tips of the fingers and also across the chest. He suffered from several like attacks, but has had none during the past year. At the time of these attacks he was using a large amount of tobacco, but has not used any during the past year. He dates his complete disability to a time when on his "beat" he was seized with a sudden, unreasoning, and uncontrollable fear—a fear that he was going to die, or something terrible was about to take place. He immediately collapsed and was taken home in a carriage, since which time he has been completely disabled. He began losing flesh, became afraid to walk on the street, was unable to ride on the street car. He waxed irritable, restless, and sleepless; hands became unsteady and trembled, gait became reeling, and heart palpitated violently on exertion, and, in fact, without exertion. His condition became so pitiable and his emaciation so extreme that the medical police official and the medical head of the Health Department in the city in which he was an officer decided that he was permanently and totally disabled. He was retired and placed on the pension roll for life.

EXAMINATION. The patient is a strongly built man, and has so far recovered his former weight that he now registers 175 pounds, instead of 130 pounds, at which weight he was retired. His complexion is florid, hair reddish-brown, and face deeply pitted from smallpox. He is extremely nervous and walks with a peculiar, uncertain, reeling gait, typically and picturesquely neurasthenic. His ears are well placed, eyes on a level, features symmetrical, tongue protruded straight, palate highly arched, with torus; palpebral aperture on the left slightly larger than the right, and the pupil of the right eye is slightly smaller than the left. There is no nystagmus, and, to rough test, no ocular palsies. The pupils react to light and accommodation and consensually. The patient states that, although his vision is good, when he reads a short time he becomes mentally confused. He is slightly short-sighted. Knee-jerks rather weak, but equal; wrist-jerks are normal; a slight jaw-jerk is present. No Romberg. Although he reels in walking, it is not a true ataxia. He appears weak in the knees. The hand grasps are interesting. The right at first registers seventy-five, second attempt sixty, third sixty, fourth fifty-five, fifth and sixth attempts fifty. After an interval of about three minutes the best he can do is fifty-five. The heart muscle is normal; pulse rapid. Thoracic and abdominal viscera normal; urinary examination negative.

Among the functional disturbances we are, perhaps, warranted in placing the isolated cases of diabetes mellitus observed and reported by Freiberg,[92] v. Frerichs,[93] and others.

The occurrence of aphasia, with or without associated paralysis as a complication and as a sequel of smallpox, has been observed.

Arnaud,[94] Combemale,[95] Saint-Phillippe,[96] Jaccoud,[97] Curschmann,[98] and Myers and Whipham[99] have reported such cases. Whether these aphasias are due, when alone, to actual destruction of the brain tissue is doubtful; that they are purely functional exhaustion or intoxication of the cortical cells of the speech centres seems more probable. Breganze's[100] case of aphasia, which occurred at the height of a variola and was complete until he made a single application of electricity, when speech returned, but remained stammering, is both puzzling and unique. He considered the loss of speech due to a cerebral atony from exhaustion of the nutrition of the cells. Notwithstanding the behavior of the speech in returning very like hysteria, we must consider the vocal halt which remained as confirmatory to his idea of its pathology, adding, perhaps, to his hypothesis the suggestion of a toxic exhaustion of the cells of the cortex presiding over emissive speech.

The following personally observed instance of aphasia in an otherwise uncomplicated case of smallpox in a boy, aged seven years, was recently reported at length:[101]

CASE IV.—E. M., white, was born in the United States, and possesses a good personal and family history. When first seen he had a temperature of 103.5° F., some coryza, a slight diarrhœa, and had vomited. There was no history of exposure to acute infectious disease, and, being informed by the parents that the child had been eating a large amount of sweets on the preceding day, I took the case to be a gastrointestinal disturbance, and treated it accordingly. On the following day the child suddenly went into violent convulsions and was more or less convulsed for an hour. The temperature was 103.8° F.; on the next day the temperature remained above 103° F., the child was stuporous, with a coated tongue and flushed face. The respiration was regular. The neck was slightly rigid, but no Kernig's sign was present. There was no rash upon the body, nor could the most careful examination disclose other signs of disease. He was thought to be in a state of meningismus of gastrointestinal origin. His condition remained unchanged, running a temperature of from 103° F., to 105° F., until the morning of the fourth day, when the characteristic eruption of smallpox appeared upon the face and calves of the legs. The eruption was discrete, frank, and classical. The temperature immediately fell and the child became conscious, but it was observed that he did not talk. He appeared to understand, but could not or would not speak. A very slight secondary fever followed, and the patient made an uninterrupted recovery. For a period of over three months the child never spoke a word. He seemed to have forgotten all the language he had ever learned. His hearing was perfect; the movements of his tongue, palate, and vocal cords were normal. He learned readily enough, and in the course of six months was speaking with almost as much fluency as before. It was impossible for obvious reasons to classify his aphasia.

BIBLIOGRAPHY.

1. Trousseau. Clinical Medicine, 1873, vol. i. p. 65.
2. Sydenham. Works of Thomas Sydenham, 1848, vol. ii. p. 252.
3. Sydenham. Quoted by Trousseau, loc. cit.
4. Berkley. Mental Diseases, New York, 1900, p. 338.
5. Kraeplin. Einfluss acuter Krankheiten, etc., Archiv f. Psych. u. Nervenkr., 1882.
6. Seppilli and Maragliano. Della Influenza del Vajuolo sulla Pazzia, Milano, 1878.
7. Trousseau. Quoted by Corlett. A Treatise on the Acute Infectious Exanthemata, Philadelphia, 1901, p. 66.
8. Corlett. A Treatise on the Acute Infectious Exanthemata, Philadelphia, 1901, p. 66.
9. Welch, Wm. M. American System of the Practice of Medicine, Philadelphia, 1897, vol. i. p. 540.
10. Westphal. Ueber eine Nervenaffektion nach Pocken, eine eigenthümliche Sprachstörung und Ataxie der Extremitäten zur Folge habend. Verhandl. d. Berl. med. Gesellsch. (1869–71), 1872, vol. iii. pp. 170–172.
11. Otto. Allgemeine Zeitsch. f. Psych., 1872–73, Band xxix. pp. 335–356.
12. Foville, S. Achille. Ann. méd. psych., 1873, p. 40.
13. Jaccoud, S. Sur un cas d'ataxie verbale suite de variole, Leçons de clinique méd, 1884–85, Paris, 1886, pp. 305–321.
14. Riva, Gaetano. Alterazioni gravi dei centri nervosi consecutive a variolo, Annali univ. di med., Milano, 1873, vol. ccxxiii. pp. 294–299.
15. Quinquaud, E. Sur quelques troubles nerveux consécutifs à la variole (fausse ataxie), l'Encéphale, Paris, 1884, pp. 33–47.
16. Leudet. Arch. gén. de méd., 1881, p. 641.

17. Whipham and Myers. Transactions of Clinical Society of London, 1886, vol. xix. p. 164.

18. Pforzheim. Schmidt's Jahrbücher, 1873, No. 1.

19. Béhier and Lionville. Sur les paralysies consécutives à quelques maladies aiguës, Thèse de Bailly, Paris, 1872.

20. Long. Landouzy, Des paralysies dans les maladies aiguës, Paris, 1880, p. 185.

21. Aldrich. Aphasia in Acute Disease, with Report of a Case Complicating Smallpox• THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1903.

22. Mabille. Démence paralytique à la suite d'une variole, Ann. méd.-psych., Paris, 1883, 6 S., vol. x. pp. 225–227.

23. Hoppe, A. Ueber Psychose nach Variolois, Greifswald, 1874.

24. Lagardelle. La variole et aliénation mentale pendant la guerre, Moulins, 1872.

25. Berthier. De l'influence de la variole sur l'aliénation mental, Gaz. méd. de Lyon, 1860, vol. xii. p. 465.

26. Kiernan, J. G. Variola and Insanity, Amer. Journ. Neurol. and Psychiat., New York, 1883, vol. ii. p. 365.

27. Gregory. Vorlesungen über die Ausschalgs-fieber (German by Helfft), Leipzig, 1845.

28. Reiner. Variola; meningitis convexitatis, Jahrb. f. Kinderk., Leipzig, 1876, x. p. 29.

29. Landouzy. Loc. cit., p. 174.

30. Landouzy. Loc. cit., p. 174.

31. Landouzy. Loc. cit., p. 177.

32. Wagner. Archiv der Heilkunde, 1872, Bd. xiii., S. 107 ff.

33. Immermann, H. Nothnagel's Encyclopedia of Practical Medicine, Amer. ed., 1902, p. 93.

34. Reiner. Variola vera; Apoplexia cerebri, loc. cit., pp. 25–27.

35. Landouzy. Loc. cit., p. 178.

36. Edes. A System of Practical Medicine, Philadelphia, 1886, vol. v.

37. Osler. Practice of Medicine, New York, 1895, 2d ed., p. 59.

38. Leroy d'Étiolles. Des Paralysies de membres inférieures, etc., Paris, 1856, T. ii. p. 93, ss.

39. Trousseau. Clinique de l'Hôtel-Dieu, T. i. p. 4.

40. Henderson, G. C. Lancet, London, December 24, 1881, p. 1089.

41. Westphal. Ueber eine Nervenaffektion nach Pocken, eine eigenthümliche Sprachstörung und Ataxia der Extremitäten zur Folge habend. Verhandl. d. Berl. med. Gesellsch. (1869–71), 1872, vol. iii. pp. 170–172; Archiv f. Psychiatrie und Nervenkrankheiten, Bd. iv. S 335 ff.; and Ueber Nerven affectionen nach Pocken, Berliner klin. Woch., 1872, No. 1; also Ueber eine Rückenmarkserkrankheit bei Paraplegie nach Pocken, ibid., No. 47.

42. Kahler and Pick. Ueber ataxie und ataxie nach acuten erkrankungen, Prager Vierteljahrschrift f. d. Prakt. Heilkund, 1879.

43. Henderson. Loc. cit.

44. Whipham. Lancet, London, December 24, 1881, p. 1089.

45. Whipham and Myers. Loc. cit.

46. Marie, P. Sclerose en plaques et maladies infectieuses, Progrès méd., 1884, vol. xii.

47. Sottas. Un cas de sclerose en plaques dans la convalescence de la variole, Gaz. des Hôp. Avril 12, 1892, p. 405.

48. Long, quoted by Gower. Diseases of the Nervous System, Philadelphia, 1893, vol. ii. p. 898.

49. Charcot, quoted by Gower. Ibid.

50. Westphal. Ueber eine Rückenmarkserkrankheit bei Paraplegie nach Pocken, Berliner klin. Woch., 1872, No. 47.

51. Damaschino, quoted by Gower, loc. cit.

52. Joffroy. Ibid.

53. Hayem. Ibid.

54. Landouzy. Loc. cit., p. 182.

55. Fiessinger. La myelite aiguë curable dans la variole, Méd. mod., Paris, 1898, vol. ix. p. 341.

56. Mackenzie, Steven. Quoted by Corlett, loc. cit., p. 67.

57. Damaschino and Roger. Recherches anatomo-pathologiques sur la paralysie spinale de l'enfance, Gaz. méd. de Paris, 1871, p. 505.

58. Landouzy. Loc. cit., p. 184.

59. Huchard. Quoted by Landouzy, loc. cit., p. 174.

60. Landouzy. Loc. cit., p. 175.

61. Marinesco and Oettinger. Semaine médicale, 1895, No. 6.

62. Gubler. Archives générales de médecine, 1860, T. i. p. 537, ss.

63. Leyden. Klinik der Rückenmarkskrankheiten, Bd. ii. S. 201, ff.

64. Gros. De quelques accidents nerveux compliquaint la variolie et la varioloïde, Alger méd., 1883, vol. xi. pp. 328, 353.

65. Chalvet. Gaz. des hôpitaux, 1871, Nr. 93: also Paralysie ascendante aiguë, Thèse de Paris, 1871,
66. Marie, P. Loc. cit.
67. Bernhardt. Berliner klin. Wochensch., 1871, No. 47, S. 561, ff.
68. Gros and Beauvais. L'Un. méd., 1894, No. 131.
69. Goss, F. W. Boston Med. and Surg. Journ., vol. lxxxviii. p. 464.
70. Friedrich, M. Personal communication.
71. Oettinger and Marinesco. Loc. cit.
72. Vulpian. Arch. de physiologie normale et pathologique, January, 1873.
73. Ebstein, W. Deuts. Arch. f. klin. Med., 1872, vol. ix. 528; vol. x. p. 595.
74. Curschmann. Wiesbadener Congress für innere Medicin, 1886, S. 469, ff.
75. Leyden. Loc. cit.
76. Combemale. Archives générales de médecine, June, 1892.
77. Curschmann. Loc. cit.
78. Putnam, James J. Nervous Disorders Occurring during Variola. Boston Med. and Surg. Journ., vol. lxxxix. p. 125.
79. Vulpian. Analyse du mémoire de Westphal, in Arch. de physiologie, 1873, p. 95.
80. Hitzig. Quoted by Putnam, loc. cit., p. 127.
81. Eulenberg. Functionelle Nervenkrankheiten, Berlin, 1871.
82. Rosenblatt, E. Porazénie miesni podudzia prawego po ospie (Paralysis of the Muscles of the Leg after Smallpox), Przegl. lek. Kraków, 1883, vol. xxii. p. 521.
83. Grocco, P. Milano, Centralbl. f. med Wissen., 1885, p. 693.
84. Gower. Diseases of Nervous System, third edition, Philadelphia, 1899, vol. i. p. 148.
85. Joffroy. Obs. II., du mémoire de Joffroy. De la névrite parenchymateuse spontanée, Arch. de Physiol., 1879, No. 2, p. 177.
86. Charcot. Amyotrophie du membre superieur droit consécutive à la Variole chez un fellah, N., incog. de la Salpêtrière, Paris, 1898, vol. xi. p. 57.
87. Rosenthal. Nervenkrankheiten, Erlangen, 1870.
88. Bierlingus. Epilepsia et paralysis ex variolis Advers. curios. cent. I., Jenæ, 1679.
89. Osler. Loc. cit.
90. Armstrong, H. E. British Medical Journal, November 11, 1871, vol. ii. p. 558.
91. Dreyfus-Brisac. Landouzy, loc. cit., p. 187.
92. Freiberg. Menschenblattern und Schutzpockenimpfung, Erlangen, 1874.
93. Frerichs. Ueber die Diabetes, Berlin, 1884, S. 222.
94. Arnaud. Troubles de la parole consécutifs a la variole, Marseille méd., 1896, vol. xxxiii. pp. 129-40.
95. Combemale. Loc. cit.
96. Saint-Phillippe. Quoted by Combemale, loc. cit.
97. Jaccoud. Loc. cit.
98. Curschmann. Loc. cit.
99. Myers and Whipham. Loc. cit.
100. Breganze. Allgem. med. Central-Zeitung, December 7, 1872.
101. Aldrich. Loc. cit.

A CASE OF TYPHOID FEVER PRESENTING AN ARTERIAL COMPLICATION—PROBABLY AN ARTERITIS.

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Among the protean complications and sequelæ of typhoid fever, those connected with the circulatory system are of great interest. They were recognized at least as long ago as 1806, when Hildenbrand,[1] in an epidemic of typhoid at Cracow, observed some cases in which

Ueber den austeckenden Typhus, etc., 8°, Wien, 1810, p. 310.